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PARTURIENT PARESIS (MILK FEVER) AND ECLAMPSIA.*

SIMILARITIES BETWEEN THESE TWO DISEASES.

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In June, 1909, the attention of one of us, Healy, was called by Dr. M. A. Scovell, director of the Kentucky Agricultural Experiment Station, to the condition known in veterinary medicine as parturient paresis. Dr. Scovell's intention was to have, if possible, the etiology of this condition definitely cleared up.

Owing to the press of other work, it was impossible to take up this problem until a year ago. As our studies progressed, the similarity between parturient paresis and that condition known in human medicine as eclampsia became more and more evident, and it is the object of this paper to point out the observations upon which we base this similarity.

Parturient paresis, then, according to the best veterinary authorities, "is a nervous disorder which develops suddenly in heavy milkers after calving. It is characterized by loss of sense, of consciousness, of muscular control, by hypothermia or by hyperthermia, convulsions, coma, and mellituria."[1]

Eclampsia, according to the best medical authorities, "is an acute toxemia, occurring in the pregnant, parturient, or puerperal woman, and is usually characterized by clonic and tonic convulsions, loss of consciousness followed by more or less prolonged coma, and presenting characteristic lesions."[2]

The following are the established facts in the etiology of parturient paresis:

The most important factor is parturition, and Hess states[3] that in 170 cases he did not observe it during, nor preceding labor, nor did he observe it later than 96 hours following labor.

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[1] Law, *Text-book of Veterinary Medicine*, 2d ed., Ithaca, N.Y., 1905.

[2] Williams, *Obstetrics*, New York, 1908.

[3] Hess, *Schw. A.*, 1905, 47, p. 279.

Next in importance to parturition comes heavy milk production. It is a disease, not only of cows, but pre-eminently of individual cows that give the most abundant milk, and particularly at that age when they have reached their greatest productiveness. It is rare or unknown in other animals.

High feeding, high condition, drying up of the milk secretion in normally heavy milkers, and easy delivery are also important points in the etiology. Constitutional predisposition is important, and an animal which has survived one attack is more liable to another.

In the etiology of eclampsia, parturition also plays the most important rôle. Here, however, there are some slight differences. Eclampsia occurs most frequently during labor, next in frequency just before labor, and least frequently just after labor. The majority of cases occur in primipara, and twin pregnancy and hydramnios are predisposing factors. Constitutional predisposition is evident in the hereditary character insisted upon by some authors. It is also of interest to note the greater mortality of cases occurring in multipara.

Briefly, the symptons of parturient paresis are a sudden appearance of discomfort, without evident reason, within a period of 12 to 96 hours following an easy parturition; loss of appetite; rumination ceases; the calf is neglected; the eyes are dull and clouded, with drooping eyelids, and reddened conjunctiva; the pulse normal and sometimes strong; the breathing excited; and the animal moans or groans. As the attack progresses, the senses are dulled and the legs become unsteady and soon give way, leaving the animal prostrate. She may make an effort to arise but is rarely able to do so. The head rests on the shoulder or upper flank. The somnolence increases, passing into complete torpor and insensibility; the reflexes are all abolished; the pulse becomes soft, small and almost imperceptible; the breathing slow and stertorous. There is constipation and retention of urine. Death may occur quietly from apoplexy, cerebral compression, or cardiac failure. On the other hand, death may be preceded by marked excitement, or disorderly muscular movements, amounting to convulsions, during which bones are frequently fractured and the animal otherwise seriously injured. In the beginning of the attack, the temperature may rise to 103° F. or 104° F., but as the condition progresses the temperature diminishes steadily until it falls below normal. With recovery, it promptly rises again to normal. In favorable cases, after the animal is down, and even without treatment, the secretion of the milk may continue, and spontaneous defecation and micturition occur and the animal may arise and commence feeding. It is of particular interest to note that the suddenness of improvement is often as marked as that of the attack. It is also of interest to note that in some animals the hind limbs remain paralytic for days or even weeks.

While the eclamptic convulsion may occur with the suddenness of "a bolt from a clear sky," it is much more common to see the outbreak preceded by a longer or shorter period of premonitory symptoms among which occur edema, headache, epigastric pains, and disturbances of vision. The convulsion is ushered in with a fixed expression of the eyes, which soon roll from side to side. The convulsive movements appear first about the mouth, the entire face becoming distorted, then extend rapidly to the arms, body and legs. They are usually clonic but may be tonic in form. The breathing is stertorous, face congested and flushed, and the mouth foams. During the convulsions the patient is unconscious, and after the convulsions cease passes into coma. There may be but a single convulsion, after which the patient makes an uninterrupted recovery. More frequently, however, there are many convulsions, the number of which varies greatly in different cases. A fatal coma may, in rare cases, follow a single convulsion. The pulse is full and bounding, but in severe cases is weaker, more rapid, becoming soft and very small. The temperature rises at the onset to 104° F. or 105° F., and steadily falls with the improvement of the patient. The temperature may, however, remain normal. Williams calls attention to the fact that there are cases in which convulsions do not occur, and states that a correct diagnosis can usually be established only by the demonstration of typical eclamptic lesions at autopsy. He further states, that in the few instances reported in which the disorder did not appear until several weeks after the birth of the child, the cases were probably not eclamptic at all, but that the seizures were due to other causes.

The urinary findings are the most important clinical features of eclampsia; there is always diminished secretion of urine, and frequently almost entire suppression. Albumen is almost always present, but this albuminuria is only temporary and rapidly disappears. The total nitrogen is diminished, as is also the urea, and the ammonia is markedly increased. Hyalin, granular and a few epithelial casts are found microscopically, as well as isolated renal cells and blood cells.

In parturient paresis, the following urinalyses of three cases will show that the urinary findings also constitute the most important clinical features. There is always diminished secretion, as well as retention of urine, the retention in one of our cases lasting 22 hours. There is always albumen present. The total nitrogen is increased. The hippuric acid is diminished, while the urea is increased. The ammonia is markedly increased. Microscopically, one finds hyalin, granular and a few epithelial casts and leukocytes.

As the result of 100 urinalyses of the dairy cow, covering a period of three winter months and three summer months, we have established the following as a normal clinical urinalysis:

Physical properties.—Colorless to deep yellow, clear, either no precipitate or a slight flocculent, or granular and occasionally a heavy white precipitate of calcium sulphate or triple phosphates. Reaction, alkaline, neutral or amphoteric, specific gravity—1,014.

Chemical properties.—Hippuric acid, 1.17 per cent; urea, 1.06 per cent; ammonia, 0.0009 per cent; total nitrogen, 0.58 per cent; no albumen; no sugar.

Microscopical examination.—Crystals of calcium sulphate, calcium oxalate, triple phosphates, ammonium urate, amorphous urate, squamous epithelial cells and irregular vegetable cells.

The following are the urinalyses of three cases of parturient paresis:

1.—Seven years, fifth calf, first attack, attack occurred 12 hours after labor. Her recovery was prompt and complete, with the oxygen treatment. Her mother died of parturient paresis.

Urinalysis.—Deep yellow, clear, white flocculent precipitate, strongly alkaline, specific gravity, 1,055; hippuric acid, 0.93 per cent; urea 3.23 per cent; ammonia, trace; total nitrogen, 1.58 per cent; abundant albumen; sugar, 6.25 per cent.

Microscopical examination.—Hyalin, granular and epithelial casts; quantities of leukocytes, squamous epithelial cells and irregular vegetable cells.

2.—Six years, fourth calf, first attack; attack occurred six hours after labor. Prompt and complete recovery, with the oxygen treatment.

Urinalysis.—Yellow, slightly cloudy, flocculent precipitate, amphoteric; specific gravity, 1,023; hippuric acid, 0.34 per cent; urea, 2.2 per cent; ammonia, 0.037 per cent; total nitrogen, 1.05 per cent; abundant albumen; sugar, 2.2 per cent.

Microscopical examination.—Numerous small granular casts, some large granular casts, and a few hyalin casts; quantities of degenerating squamous and cuboidal epithelial cells, leukocytes and irregular vegetable cells.

3.—Seven years, fifth calf, first attack, attack occurred 36 hours after labor. Recovery prompt and complete, with the oxygen treatment.

Urinalysis.—Yellow, clear, slight flocculent precipitate, slightly acid; specific gravity, 1,018; hippuric acid, 0.83 per cent; urea, 1.20 per cent; ammonia, 0.035 per cent; total nitrogen, 0.65 per cent; moderate amount of albumen present, sugar 1.64 per cent.

Microscopical examination.—Granular and hyalin casts, squamous epithelia and leukocytes, irregular vegetable cells.

The characteristic lesions found at autopsy in cases of eclampsia are areas of necrosis, in which blood cells may or may not be present in the liver, in the neighborhood of the smaller portal vessels; that is a hemorrhagic hepatitis. There is an acute parenchymatous degeneration of the kidneys, with but little disturbance of the glomerule and interstitial substances. There is also a coagulation necrosis, either focal or diffuse, of the adrenals, and pneumonia is not infrequent.[1]

Unfortunately, the finer pathological changes occurring in parturient paresis have not been established, and none of the three cases which we had during the past year died. Nevertheless, we shall show in our second paper, that three guinea-pigs which died under the influence of the toxin of parturient paresis in five, six, and seven days, presented upon microscopical examination, areas of hemorrhagic necrosis in the liver, an acute parenchymatous nephritis with interstitial hemorrhage, and an acute degeneration of the adrenals with interstitial hemorrhage; and, in one case, a left lobar pneumonia. That in another guinea-pig, which received a smaller amount of the toxin, the immediate result was an intense diuresis, accompanied with an albuminuria, the animal passing more than 200 c.c. of urine in the 24 hours. This animal apparently recovered, and 10 days later was chloroformed. Upon microscopical examination, we found an acute parenchymatous nephritis, with some interstitial hemorrhage; a rather extensive necrosis of the liver cells but without hemorrhage; and localized areas of necrosis in the adrenals.

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[1] Adami and Nichols, *Principles of Pathology*, 2, 1911. (This contains the best description of the kidney of pregnancy which we have seen.)

We now come to the most interesting part of this whole problem, namely, the cause underlying, on the one hand, eclampsia, and on the other, parturient paresis.

As Zweifel has aptly put it, "Eclampsia is the disease of theories." It has been identified with uremia; it has been held that it was the result of anemia and edema of the brain; of the circulation of ammonium carbonate in the blood; that it was the result of bacterial invasion; of the circulation of carbamic acid in the blood; that it was due to poisons arising from the fetal elements; that it was due to poisons absorbed from the placenta; that it was due to thrombosis, caused by placental cells passing into the maternal circulation; that it was due to the circulation of lactic acid in the blood; that it was due to thyroid insufficiency; and again to irregularities in the metabolism of the parathyroid bodies; and, lastly, that it was due to autointoxication. There is not sufficient experimental evidence, however, to establish any of the above theories, and it is of interest to note that the possibility of the breast elaborating the toxin has not, as yet, been investigated.

Parturient paresis has also been explained by many theories. It was considered the result of emotional excitement; of cerebral anemia; of cardiac hypertrophy; of bacterial infection; as the reaction of surplus nerve force, following an easy labor; and of an increased percentage of sugar in the blood. The most remarkable feature presented by parturient paresis is the rapid response to, and the entire success of, the modern treatment. This treatment has reduced the mortality from 60 per cent to 0.5 per cent, and the rapidity of recovery is very striking indeed. An animal, apparently in the last stage of coma, will often within a few hours, or even within twenty minutes, after the treatment, be up and eating and licking her calf, apparently well and contented. The treatment consists of acute dilatation of the udder, by means of suitable liquids or gases. The treatment was first introduced by J. Schmidt of Kolding, Denmark, and was based upon the theory that the disease was due to a bacterial infection of the udder. He used for injections into the udder solutions of potassium iodid, but it has since been shown that solutions of lysol, or of sodium chlorid, will act as well, and that the injections of sterile water, sterile air, and of oxygen are even more successful. Injections of oxygen or of sterile air now constitute the generally used treatment.

That the disease is due to a toxin elaborated in the udder, as the result of its own metabolism preceding normal milk production, there can be no reasonable doubt, and that the success of the modern treatment is due to preventing, by means of pressure, the absorption of this toxin seems most highly probable.

We are of the opinion that eclampsia is due to a similar toxin elaborated by the breast in a similar manner, and would strongly recommend, as the most promising treatment, dilatation of the breasts with oxygen or sterile air, accompanied by vigorous massage of the breasts, or forcible compression of them by means of a properly applied bandage, at the same time using whatever medical measures may be indicated. We ourselves shall thoroughly test this method of treatment as soon as the opportunity occurs.